THE UNIVERSITY OF ALBERTA

EFFECTS OF ENDOTRACHEAL SUCTION AND LUNG HYPERINFLATION ON ARTERIAL OXYGENATION IN CRITICALLY ILL ADULTS

by

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Dedication

We give thee but Thine own,
What - e'er the gift may be:
All that we have is Thine alone,
A trust, O Lord, from Thee.

Hymn (William How)

There is nothing for anyone to boast of.
For we are God's handiwork, created in
Christ Jesus to devote ourselves in the
good deeds for which God has designed us.

Ephesians 2:10

If a man must boast, let him boast of the Lord.

First Corinthians 1:31

Abstract

Endotracheal suctioning is frequently required to remove pulmonary secretions from the respiratory tracts of critically ill patients. The suction procedure however, may place the patient at risk for hypoxemia. The purpose of this study, therefore, was to assess the efficacies of four endotracheal suction protocols in reducing or preventing suction-induced hypoxemia.

The sample for this study consisted of 24 intubated and ventilated, critically ill adult patients. A two-way (Protocol x Time) within subjects counterbalanced experimental design was used. Each subject was exposed to all four suction protocols and during each protocol, dependent variables were measured at six time intervals (prebaseline, baseline, and 0, 60, 120, 180 seconds after suctioning). Therefore, 24 measures were obtained on each dependent variable for each subject.

Suction Protocol I was the hospital ICU suction procedure, which reflected standard criteria for suctioning. Protocols II, III and IV were identical to Protocol I except for the addition of hyperinflations. The time of administration of the hyperinflations differed for Protocols II to IV. Protocol II hyperinflations occurred before suctioning; Protocol III hyperinflations occurred after suctioning; and Protocol IV hyperinflations occurred before, between and after suctioning. A hyperinflation was defined at 1.5 times the tidal volume setting on the

patient's volume control ventilator. The following dependent variables were of interest: heart rate; partial pressure of oxygen ($Pa0_2$); saturation of oxygen ($Sa0_2$) calculated from $Pa0_2$ and blood pH as well as measured directly with an ear oximeter; and the transcutaneous partial pressure of oxygen ($tcP0_2$).

Analysis of $Pa0_2$ data demonstrated significant Protocol and Time main effects and a significant Protocol x Time interaction. Significant Time effect and Protocol x Time interaction were also found for calculated $Sa0_2$. These data indicated that Protocol IV was most effective in guarding against suction-induced hypoxemia since $Pa0_2$ was high compared to the other protocols. The real value of the hyperinflation procedure in Protocol IV seemed to be the repeated hyperinflations (before, between and after suctioning). Heart rate generally was not sensitive to the same effects.

Other analyses demonstrated high positive correlations between calculated $Sa0_2$ and ear oximeter $Sa0_2$ and between $Pa0_2$ and $tcP0_2$.

In this study the implications of hyperinflations in protecting the patient from suction-induced hypoxemia were discussed and suggestions regarding nursing practice were offered.

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Special mention belongs to our family and friends who not only supported us with prayer (as we both completed masters degrees) but also, in very practical ways, demonstrated their caring and helped us retain our sense of perspective.

And finally, to my husband who says, "Aren't you going to mention my name?"....Hello, Tony!"

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I. Introduction

Endotracheal suctioning is frequently required to remove pulmonary secretions from the respiratory tracts of critically ill patients. In the intensive care unit (I.C.U.) it is the nurse who is responsible for: (1) assessing the need for endotracheal suctioning, (2) executing the procedure, and (3) evaluating the outcome (Grossbach-Landis, 1979). Since suctioning removes air (oxygen) as well as secretions it should not be regarded as a benign nursing procedure.

When the partial pressure of oxygen (PaO_2) in the arterial blood falls well below normal, as it can with suctioning, complications such as cardiac arrhythmias and death may occur (Shim, Fine, Fernandez & Williams, 1967; Sloan, 1950). Those patients at risk for the untoward effects of suctioning include those who have: (1) a PaO_2 less than 70 mm Hg and, (2) those who are in generally poor condition (hypotension, acid-base imbalance, arrhythmias)(Jacquette, 1971).

Using these risk criteria, it is apparent that many I.C.U. patients would qualify as "high risk" candidates for the untoward effects of suctioning. Taylor and Waters' (1971) study supports this notion as patients with cardiovascular and/or respiratory abnormalities experienced significantly greater drops in PaO_2 with suctioning than those with normal cardiopulmonary systems.

On the basis of research several guidelines regarding suctioning have been implemented into nursing practice. These include aseptic technique (Demers & Saklad, 1973), duration of suctioning (Boutros, 1970), ratio of the external diameter of the suction catheter to the internal diameter of the endotracheal tube (Rosen & Hillard, 1962) and preoxygenation of the patient (Harken, 1975). However, studies on suction procedure variables have failed to pinpoint one specific suction method that consistently prevents or minimizes suction-induced hypoxemia (the decrease in PaO_2 associated with suctioning). Hence, a variety of suction procedures are evident in the clinical setting today.

The range of options available to nurses at the bedside can be observed in the various methods used to oxygenate patients prior to suctioning. These include hyperventilation, bagging, hyperinflation, sighs and increasing the fractional concentration of oxygen (FIO_2).

> Furthermore all combinations of these manoevers are practiced without assessment of their effectiveness. There is little doubt that the endotracheal suctioning procedure in critically ill patients needs to be examined in well-designed research studies (Adlkofer, 1978, p. 1014).

A. Purpose of the Study

The focus of this study was suction-induced hypoxemia. The purpose of this study was to investigate the effectiveness of endotracheal suction protocols in reducing or preventing suction-induced hypoxemia in intubated I.C.U.

patients. The questions of interest were: (1) Would hyperinflations prevent or minimize suction-induced hypoxemia? (2) If so, would it make a difference when the hyperinflations were delivered (before, after or throughout suctioning)?

Several of the terms appearing in the literature review are defined below.

B. Definition of Terms

1. Endotracheal suctioning refers to a routine nursing procedure executed on intubated patients. It involves passing a suction catheter into an oral or nasal endotracheal tube or a tracheostomy tube for the purpose of aspirating secretions thereby maintaining airway patency.
2. Hypoxemia refers to a decreased oxygen tension in the blood (Hunter, 1981). A PaO_2 of 55 to 60 mm Hg is often used as a cut off value for treatment of hypoxemia (Rokosky, 1981).
3. Hypoxia refers to a deficiency of oxygen in a nonspecified area, that is, tissue hypoxia, organ hypoxia, or hypoxia generalized to the entire organism (Brannin, 1974).
4. Arterial line refers to an indwelling catheter inserted in an artery for the purpose of obtaining blood samples conveniently, without the need for frequent arterial

puncture.

5. Arterial blood gas (ABG) refers to the analysis of a sample of heparinized arterial blood for PaO_2, the partial pressure of carbon dioxide ($PaCO_2$) and the pH. The saturation of arterial blood with O_2(SaO_2) will be calculated from PaO_2, pH and patient temperature.
6. Saturation of oxygen (SaO_2) refers to the percentage of oxygen bound to hemoglobin (Hunter, 1981). An oxygen saturation of 90 to 93% represents a critical range below which severe arterial hypoxemia may rapidly occur because of the steep slope of the oxyhemoglobin dissociation curve below this range (Downes & Wilson, 1961).
7. FIO_2 refers to the fractional concentration of inspired oxygen. Room air has an FIO_2 of 0.21.
8. Tidal volume (VT) refers to the volume of gas inspired or expired during the normal respiratory cycle. Normal tidal volume is calculated to be 5 to 7 ml/kg (Hunter, 1981). In acutely ill ventilated patients the VT is often 10 ml/kg or more.
9. Alveolar-arterial gradient ($AaDO_2$) refers to the difference between the oxygen tension of the alveolar air and the PaO_2 once respiration has taken place (Jacquette, 1971).
10. Right-to-left shunting refers to a condition that occurs when there are large areas of the lung that are not ventilated (because of atelectasis, pulmonary edema

etc.) and yet continue to have normal blood flow. Therefore blood leaving the lung through the pulmonary veins has an O_2-content the same as that in the pulmonary arteries (Robertson & Guzzetta, 1976).

II. Review of the Literature

Research findings related to the focus of this study are discussed in this chapter. The two main areas of review concern the physiological sequelae of endotracheal suctioning (the hypoxemic response, suction and the hypoxemic response, suction and trauma) and suction procedure variables, specifically hyperinflations, which are thought to minimize the adverse effects of suctioning.

A. Physiological Sequelae of Endotracheal Suctioning

The literature indicates that tracheal suction results in hypoxemia thereby exposing the critically ill patient to certain risks as well as benefits. The following review of the sequelae of endotracheal suctioning is summarized graphically in Figure 1.

The Hypoxemic Response

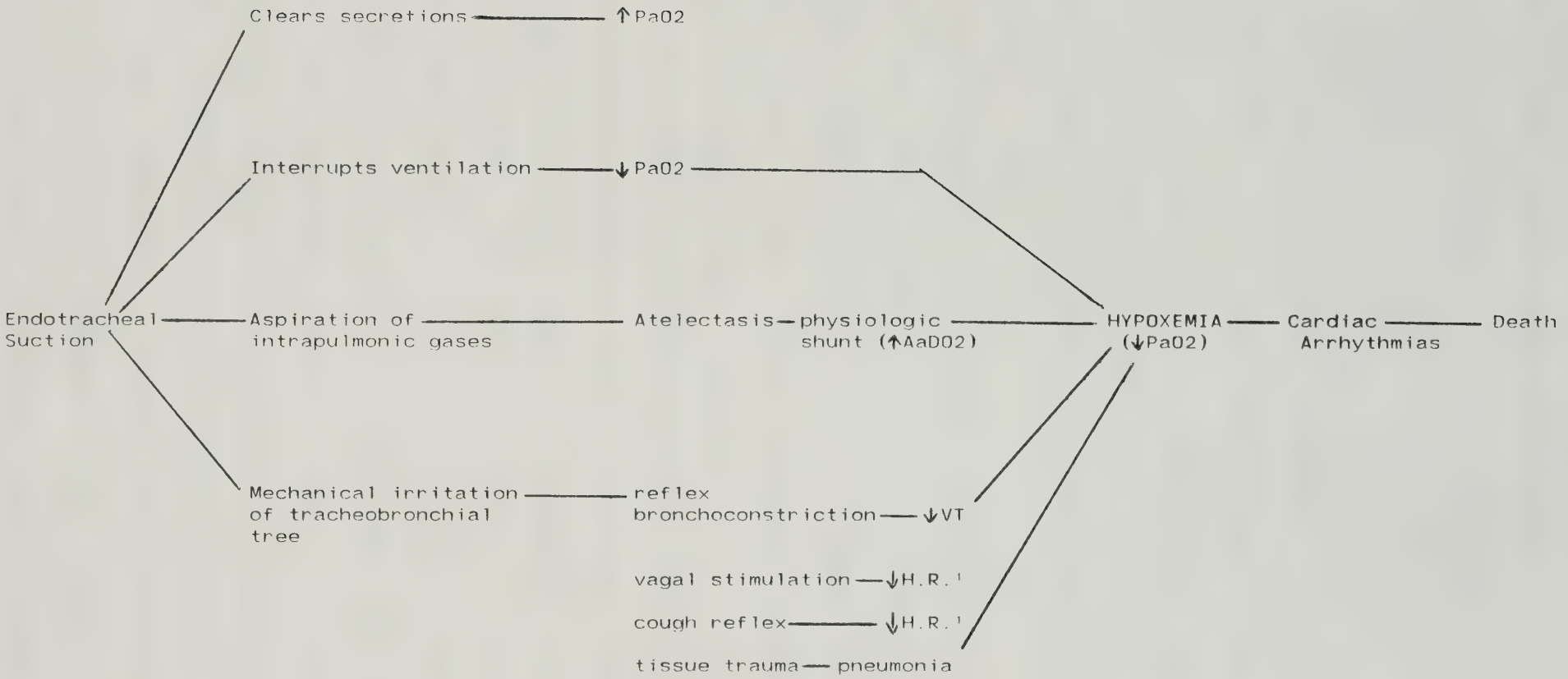
The removal of intrapulmonic gases during suctioning results in alveolar hypoxia and a subsequent fall in arterial oxygen tension. The chemoreceptors in the carotid and aortic bodies, sensing a drop in PaO_2, respond by increasing the depth and frequency of respiration (Mountcastle, 1974). This reflex serves to rid the body of CO_2. Pulmonary vasoconstriction will occur in response to a decrease in alveolar oxygen tension (PAO_2) thereby restricting perfusion to unventilated lung segments and increasing pulmonary artery pressure (Pirlo, Benumof &

Trousdale, 1981; Simbruner, Coradello, Fodor, Havelec, Lubec & Pollak, 1981).

In the peripheral circulation however, there occurs a vasodilation as the body attempts to deliver available oxygen to the tissues. It has been observed also that more pulmonary capillaries are perfused during hypoxia than normoxia. The reason for this is that airway hypoxia causes an increase in pulmonary artery pressure which in turn causes pulmonary capillary recruitment for the purpose of increasing the gas exchange surface (Capen, Latham & Wagner, 1981).

Once the chemoreceptors have been stimulated, epinephrine is released by the sympathetic nervous system causing an increase in heart rate, blood pressure and cardiac output (Brannin, 1974; Mountcastle, 1974). Dripps and Comroe (1947) found that an increase in pulse rate was a more sensitive index of hypoxemia than an increase in respiratory minute volume.

If hypoxemia persists systemic hypoxia may ensue (Demers, 1982). Cerebral hypoxia will result in progressive depression of central nervous system functions, namely; dizziness, euphoria, sensory disturbances, muscular weakness and ultimately unconsciousness (Mouncastle, 1974). Myocardial hypoxia increases cardiac irritability and causes a resetting of the natural pacemaker (premature contractions, nodal rhythms). Such arrhythmias may develop into bradycardia and asystole (Jacquette, 1971).



[1] heart rate

Figure 1. The sequelae of endotracheal suctioning.

Suction and the Hypoxemic Response

> Since secretions block alveolar ventilation and promote shunting of venous blood into the arterial system, a rise in arterial PO_2 should follow the removal of mucous. However, when the conventional suctioning techniques are used, arterial PO_2 is lower during and after suctioning than it is at the pre-suction levels (Jacquette, 1971, p. 2362).

Studies of humans have found a mean drop in PaO_2 from pre-suction levels of 35 mm Hg during suction (Bodai, 1982), 38 mm Hg immediately post-suction (Urban & Weitener, 1969), and 33 mm Hg thirty seconds post-suction (Skelley, Deeren & Powaser, 1980).

As expected there was a concomitant fall in SaO_2 with a drop in PaO_2 (Boba, Cincotti, Piazza & Landmesser, 1959; Kergin, Bean & Paul, 1948). These data establish that suctioning does indeed disrupt ventilation and induce alveolar hypoxia and hypoxemia.

Several authors have documented sustained falls in PaO_2 associated with suctioning. Skelley, Deeren and Powaser (1980) along with Taylor and Waters (1971) noted that three minutes after suctioning, their subjects' PaO_2 still remained below pre-suction values. This occurred despite the fact that subjects were mechanically ventilated during the post-suction period. Similar research done on dogs indicated a fall in PaO_2 persisting up to at least 5 minutes post-suctioning (Naigow & Powaser, 1977).

The mechanism behind this prolonged drop in PaO_2 remains a matter of speculation. Some authors suggest that PaO_2 values remain low after suctioning because the negative

airway pressure gives rise to atelectasis (Rosen & Hillard, 1960, 1962). Brandstater and Muallem (1969) reported that suctioning of newborns caused areas of lung collapse which persisted for up to thirty minutes. Mechanical ventilation for 30 minutes, at pre-suction settings, did not reinflate atelectic areas post-suction.

> What happens to a collapsed lung which is not reinflated is a matter of conjecture. Some areas may open slowly over a period of hours unless repeated suction adds injury to insult. Other areas may remain collapsed, and the next likely event is infection and pneumonia (Brandstater & Muallem, 1969, p. 472).

Those who refuted the position that negative pressure is responsible for the fall in PaO_2 have cited studies comparing apnea with suction (Boba, Cincotti, Piazza & Landmesser, 1959; Downes, Wilson & Goodson, 1961; Ehrhart, Hofman & Loveland, 1981). These researchers found no significant difference between comparable periods of apnea and suction with respect to SaO_2 and PaO_2. Boutros (1970) however documented that ten seconds of suction did result in a significantly greater drop in PaO_2 than ten seconds of apnea.

It has been suggested that the discrepancy between these findings may be attributed to the sensitivity of oxygen measures monitored in the four studies. Boutros (1970) and Ehrhart et al. (1981) measured PaO_2 wheras Boba et al. (1959) and Downes et al. (1961) measured SaO_2 which will change very little as PaO_2 decreases from 100 to 60 mm Hg. Differences among the findings might also be attributed

to suction catheter size. Downes et al. (1961) did not report catheter size but in the study done by Boba et al. (1959) the ratio of the inner diameter of the endotracheal tube to the outer diameter of the suction catheter was 2.1:1 compared with 2:1 in the study by Ehrhart et al. (1981) and 1.8:1 in the study by Boutros (1970).

> The average subatmospheric pressure developed in the lungs during suction increases as this ratio decreases and the development of a more subatmospheric pressure increases the liklihood of atelectasis and reduction of total O_2 volume in the lungs (Ehrhart, Hofman & Loveland, 1981).

Investigations into the mechanisms responsible for the sustained fall in PaO_2 after suctioning have yielded some interesting results. It has been shown that insertion of a catheter alone, without suction, will cause a drop in PaO_2 and SaO_2 similar to that seen when suction is applied (Boba, Cincotti, Piazza & Landmesser, 1959; Woodburne & Powaser, 1980). These authors attributed their findings to respiratory reflexes.

Mechanical irritation of the tracheobronchial tree by a suction catheter may stimulate vagal receptors. The cough reflex is entirely vagal and is accompanied by broncho-constriction, bradycardia and a transient rise in intrathoracic pressure (Demers, 1982; Harken, 1975; Mountcastle, 1974). Research done on paralyzed artificially ventilated cats indicated that stimulation of the laryngeal tracheobronchial regions causes an increase in total lung resistance (Nadel & Widdicombe, 1962), reflex tracheo-bronchoconstriction and an increase in systemic

blood pressure (Tomori & Widdicombe, 1969). In man, as in cats, stimulation of the lower airway produces an arterial pressor response and tachycardia (Corbett, Kerr, & Prys-Roberts, 1968).

Reflexes related to tracheal stimulation and enhanced by hypoxemia may result in cardiac arrhythmias. There are several reports of sudden cardiac death during a period of suctioning reported in the literature (Keown, 1960; Marx, Steen & Arkins, 1968; Schumaker & Hampton, 1951). The vagal effect by itself however does not cause cardiac arrest. It appears that some degree of hypoxia must be present along with vagal stimulation (Ayres & Grace, 1969; Sloan, 1950).

Katz and associates (1981) measured SaO_2 and monitored the electrocardiograms of fifty patients during fiberoptic bronchoscopy. Forty percent of the subjects developed major disturbances of cardiac rhythm and these arrhythmias were most frequently associated with periods of maximum oxygen desaturation. In another study the incidence of transient cardiac arrhythmias was significant during tracheal suctioning while the patients were breathing room air. However, when these same individuals were given 100% oxygen prior to the suction procedure no arrhythmias occured (Shim, Fine, Fernandez & Williams, 1969).

Suction and Trauma

As the amount of negative pressure applied during suction increases so too does the damage done to the

respiratory tract. Kuzenski (1978) suctioned two dogs once every fifteen minutes over a period of four hours. The first dog was suctioned with -100 mm Hg pressure and on autopsy the trachea showed areas of denuded epithelium, leukocytic infiltration and edema. The trachea of the second dog, suctioned at -200 mm Hg, indicated more extensive damage including loss of cilia and submucosal involvement.

Destruction of ciliated epithelium by the suction catheter suppresses mucous clearance thereby predisposing the tracheobronchial tree to infection (Sackner, Landa, Greeneltch & Robinson, 1973). Damage to the mucous glands in the submucosa will cause a decrease in mucous secretion and diminished protection from tracheobronchial irritants (Kuzenski, 1978).

In one study, conducted on kittens, it was demonstrated that tracheal ulceration occurred three hours after suction commenced (Thambiran, 1966). Trauma was also evident with catheter insertion alone and with suction pressures as low as 50 mm Hg. Hilding (1964) has shown that light stroking of extirpated trachea with a cotton swab causes complete exfoliation of the columnar layer over a period of 1 to 24 hours.

In clinical practice trauma would be expected to be greater than in the laboratory studies because intubated patients are subjected to frequent suctioning for periods ranging from days to weeks. After evaluating four suction

catheters, of different design, Jung and Gottlieb (1976) concluded that "mucosal trauma with suctioning procedures is more likely due to repetition, vigor, and amount of suction applied, regardless of which type of catheter is used" (p. 179).

B. Suction Procedure Variables

Several factors have been thought to influence the degree of hypoxemia induced by endotracheal suctioning, namely:

1. the duration of applied suction (Boutros, 1970; Woodburne & Powaser, 1980);
2. the magnitude of suction pressure and flow (Kuzenski, 1978; Rosen & Hillard, 1960, 1962; Rux & Powaser, 1979; Thambiran & Ripley, 1966);
3. the ratio of suction catheter size to endotracheal tube size (Demers & Saklad, 1973; Rosen & Hillard, 1960, 1962);
4. the design of the suction catheter (Jung & Gottlieb, 1976; Landa, Chapman & Sackner, 1980; Link, Spaeth, Wahle, Penny & Glover, 1976; Sackner, Landa, Greeneltch & Robinson, 1973);
5. continuous versus intermittent suction (Deeren & Powaser, 1979);
6. suctioning with continuous oxygen insufflation (Belling, Kelly & Simon, 1978; Berman & Stahl, 1968; Bodai, 1982; Boba, Cincotti, Piazza, Landmesser, 1959; Fell & Cheney,

1971; Jung & Newman, 1982; Langrehr, Washburn & Guthrie, 1981; Powaser, Langrehr & Cambell, 1979; Urban & Weitzner, 1969).

7. suctioning with high frequency jet ventilation (Keszler, 1980; Spoerel & Chan, 1976);
8. oxygenation of the patient prior to suctioning (Adlkofer & Powaser, 1978; Boutros, 1970; Naigow & Powaser, 1977; Weitzner, King & Ikezeno, 1959).

Based on the findings and recommendations of the above research, a suction procedure should include; (1) Increasing the FIO_2 several minutes prior to suctioning (100% O_2 is often suggested). (2) Using aseptic technique throughout the procedure (sterile gloves, mask and suction catheter). (3) Choosing a transparent suction catheter with multiple side holes and an external diameter no more than half the internal diameter of the endotracheal tube. (4) Suctioning for fifteen seconds or less. (5) Avoidance of high negative pressures by using the minimum amount of suction pressure that will clear the airway. (6) Hyperinflation of the lungs prior to and/or after suctioning. (7) Allowing the patient to catch his breath between consecutive suction passes (bag or reconnect patient to the ventilator). (8) Insertion of the catheter (with the side bore open) until resistance is met then withdrawing the catheter slightly before applying intermittent suction. (9) Suctioning no more often than is necessary.

Hyperinflation

It is evident from the literature that hyperinflations are an important adjunct to the suction procedure. It has been shown that preoxygenation with an increased FIO_2 and lung hyperinflation will prevent or at least minimize the fall in PaO_2 associated with suctioning (Demers & Saklad, 1973; Downes, Wilson & Goodson, 1961). Fell and Cheney (1971) studied patients with varying degrees of respiratory failure and found that hyperinflations with 100% oxygen for one minute prior to suction resulted in only 5% of the subjects having a PaO_2 less than 65 mm Hg immediately after suctioning. Repeating the experiment without the hyperinflations and oxygen, 39% of the same subjects had a drop in PaO_2 below 65 mm Hg following the procedure. The procedure used to deliver hyperinflations was not described in this study.

Skelley, Deeren and Powaser (1980) reported that eleven cardiac patients suctioned without preoxygenation experienced a maximum fall in PaO_2 of 33 mm Hg (mean) thirty seconds post-suctioning. However, one or three hyperinflations with 100% oxygen administered to these same patients before suctioning either minimized or totally prevented the short term fall in PaO_2. A hyperinflation in this study was defined as 1.5 x VT.

A study using two dogs revealed that three minutes of hyperinflations with 100% oxygen prior to suction did not make a significant difference regarding PaO_2 when compared

to three minutes of oxygen (100%) delivered via normal tidal volumes (Naigow & Powaser, 1977). Here hyperinflations were defined as 10 breaths/minute at volumes of 300 to 400 ml/breath. The findings from a similar study indicated that three hyperinflations with room air before suctioning failed to prevent a sustained fall in PaO_2 (Woodburne & Powaser, 1980). In this study the term hyperinflation was not operationally defined.

Brandstater and Muallen (1969) showed that a collapsed lung (due to suctioning) will remain collapsed unless high pressure or volumes are used for inflation after suctioning. Boutros (1970) studied twenty-two anesthetized patients with no pulmonary disease and found that hyperinflations "...sustained for ten seconds immediately after suctioning" resulted in significantly smaller decreases in PaO_2 than when no hyperinflations were given. Again no clear definition of the hyperinflation procedure was provided.

In the Naigow and Powaser study (with dogs as subjects) hyperinflations delivered post-suctioning (FIO_2 of 0.21) restored PaO_2 values above the control levels quickly. When hyperinflations (FIO_2 of 1.0) were delivered throughout the suction procedure a PaO_2 significantly above the control values was maintained.

The literature reveals three main problem areas in evaluating the effects of hyperinflations on suction-induced hypoxemia. First, of the handful of studies done, only two have been conducted on intubated patients with respiratory

disorders. Second, many of the studies do not include a definition of the term hyperinflation and those that do differ in their interpretation and operationalization of this term. Third, the research to date has not separated the effect of oxygen from that of the hyperinflations that is, hyperinflations are often delivered with 100% oxygen. In short, the validity of "conventional" suction procedures incorporating hyperinflations has not been established.

In this study it is hypothesized that hyperinflations, included in the suction procedure, will minimize suction-induced hypoxemia. More specific details of this hypothesis will be provided after the study procedure is presented.

III. Methods

A. Design

A two-way within subjects experimental design was used for this investigation. Each subject was tested on four separate occasions and on each occasion a different suction protocol was evaluated. In addition, during each protocol physiologic parameters were recorded several times for each subject. Hence the two repeated factors in this study were suction protocol and time.

The sequence of the protocols tested was controlled through use of an incomplete counterbalancing technique.

> Incomplete counterbalancing derives its name from the fact that all possible sequences of treatment conditions are not enumerated. The criterion that incomplete counterbalancing must meet is that, for the sequences enumerated, each treatment condition must appear an equal number of times in each ordinal position. Also each treatment condition must precede and be followed by every other condition an equal number of times.
>
> (Christenson, 1980, p. 138)

Since there were four treatment conditions to be evaluated in this study the total number of subjects required would be some multiple of four. It was decided that a minimum of 24 subjects were necessary to satisfy requirements for adequate statistical power. Table 1 summarizes the sequence of suction protocols for the 24 participants.

Table 1. Random assignment of suction protocol sequences for the 24 study subjects

Subject Number	Protocol Sequence
1 5 9 13 17 21	3 4 2 1
2 6 10 14 18 22	1 2 4 3
3 7 11 15 19 23	4 1 3 2
4 8 12 16 20 24	2 3 1 4

B. Sample

This study was conducted in the eleven bed Multi-System Failure I.C.U. at the University of Alberta Hospital (Edmonton, Alberta). The patients admitted to this unit were critically ill and their illnesses usually involved more than one body system. Nursing care was given on a one to one basis as the majority of patients were on respirators. Data were collected over a nine week period between 0700 to 2400 hours and subjects who consented to participate were studied over a 12 to 24 hour period.

The first 24 patients on the unit meeting the criteria for selection and agreeing to participate were chosen for the sample. All subjects selected for this study met the following criteria:

1. 16 years of age or older,
2. fluent in English,

3. conscious and in stable condition: no active bleeding, no seizures, no hemodialysis, hemoglobin ≥ 10 gm/dl., blood pressure ≥ 100/60 mm Hg,
4. attached to a cardiac monitor and also had an arterial catheter,
5. intubated (oral, nasal, tracheostomy) and ventilated by a volume control respirator,
6. on an FIO2 ≤ 0.6, VT ≤ 1,000 ml and positive end expiratory pressure (PEEP) ≤ 5cm H_2O,
7. suctioned on at least three prior occasions.

C. Ethical Considerations

Individuals meeting the selection criteria gave their written consent prior to being included in the study (Appendix I). The purpose of the study was explained to each patient in the presence of a witness (usually the patient's nurse). The patients were shown the equipment (ear oximeter and $tcPO_2$ monitor) and given the opportunity to write down any questions. It was stressed that participation was voluntary and that the patient could withdraw from the study at any time. Only one of the patients approached for consent chose not to participate.

The procedure of the study assured protection of the subject. Several blood samples (40 ml) were drawn painlessly from the patient's arterial line during each study session. Suctioning was not to be done unnecessarily because of participation in this study. Data were collected

only when the patient's nurse deemed that suctioning was required.

D. Procedure

Assembling the Equipment

The $tcPO_2$ electrode was heated to 44°C and then a 2 point gas calibration was done. The electrode was applied to the patient's chest and readings were allowed to stabilize before the actual study commenced. This took about twenty minutes. The sensor location was changed every 4-6 hours to prevent burning and the electrode was recalibrated at these times. The ear oximeter took about five minutes to warm up at which time the ear piece was calibrated and subsequently attached to the patient's ear. An elastic head band was used to keep the ear piece in proper position.

All patients were put on Assist-Control ventilation mode and removed from PEEP one half hour prior to data collection. The Assist-Control mode guaranteed that each time the patient took a breath the ventilator would deliver a pre-set tidal volume. Tidal volume and back up respiratory rates were individually set and no attempt at control was made during the present study. None of the patients in this study were receiving ventilator sighs. Subjects were removed from PEEP because it has been suggested that high levels of PEEP may serve the same

function as hyperinflation (Balsys, Jones, Man & Wells, 1980).

In all cases a size 14 French Gentle-Flo catheter (American Hospital Supply) was used to do the suctioning. The external diameter of this catheter was 4 mm. For patients with size 7 mm and 8 mm endotracheal tubes the ratio of the internal diameter of the tube to the external diameter of the catheter was 1.75:1 and 2:1 respectively. A six foot length of transparent connecting tube (Argyle) was attached to the wall suction unit and the same suction gauge (Medigas) was used for each patient. The gauge was adjusted at every testing to attain a peak suction pressure of -180 mm Hg after 5 seconds of occluding the connecting tube distal to the gauge. The suction flow rate at -180 mm Hg was 25 liters/minute.

Training of Assistants

Once the equipment was prepared, assistants were trained to help in data collection. There were two staff members involved in each data collection session. One was responsible for suctioning the patient while the other recorded the various physiologic parameters (heart rate, transcutaneous partial pressure of oxygen ($tcPO_2$) and SaO_2 from the ear oximeter). The researcher drew the ABGs, changed ventilator settings (VT, FIO_2) and supervised the whole procedure.

Available qualified I.C.U. staff members were selected to do the suctioning (nurses, respiratory technologists and physiotherapists). Note that due to the critical care setting the same staff members were often not able to participate in all data collection sessions for a particular patient. Prior to each testing the researcher reviewed the suction technique to be employed with the assistants. They were instructed to insert the catheter until resistance was met and then draw back one inch before applying continuous suction.The catheter was to be rotated through 360° while withdrawing.

The instructions for each suction protocol were put on separate magnetic cassettes. Each phase of the procedure was timed and verbal countdowns on the tapes indicated when an action was to commence and finish. The same portable tape recorder was used at every testing. The use of tape recordings ensured that suction was consistently applied and that the patients were off the ventilator for the same length of time during each protocol.

The instructions heard on the tape recording were explained to both assistants prior to each data collection session. The staff person responsible for recording the various parameters was instructed to copy the appropriate data each time a blood gas sample was drawn. Direct supervision of the recorder and the suctioner during data collection was done by the researcher.

Protocols

Four suction protocols were evaluated in this study. Protocol I, based on the University of Alberta Hospital's procedure for suctioning included: (1) increasing the FIO_2 by 0.2 prior to suctioning, (2) using sterile technique (sterile gloves, mask and catheter), (3) applying continuous suction while withdrawing the catheter, and (4) returning the patient to the ventilator after the catheter was withdrawn.

A time limit was put on each phase of the suction procedure. The FIO_2 was increased by 0.2 fourteen minutes before baseline measurements were made allowing the blood gases to equilibrate at the new oxygen concentration before testing began. In a study conducted on 92 critically ill patients it was found that PaO_2 reached 95% of its equilibrium value two minutes after the FIO_2 was changed (Tremper & Shoemaker, 1981).

Seven seconds were allotted for disconnecting the patient from the ventilator and inserting the catheter. Continuous suction was applied for 5 seconds and then 3 seconds were allowed for reconnecting the patient to the ventilator. In all, the patient was off the ventilator for 15 seconds but only suctioned for five. During each protocol the patient was suctioned three times and 30 seconds of mechanical ventilation was provided between the three suction passes.

Prior to setting these time limits several different I.C.U. nurses were timed, unbeknown to them, as they suctioned patients. The timing for each phase of the suction procedure therefore reflects the current suction practice in the University of Alberta Hospital's I.C.U.

Patients were not instilled (injection of saline into the endotracheal tube) during suctioning nor was chest physiotherapy given within one half hour before testing as both of these activities could influence arterial oxygenation (Connors, Hammon, Martin & Rogers, 1980). During each testing patients were supine with their heads midline.

Protocols II, III and IV were identical to Protocol I except for the addition of hyperinflations. Hyperinflations were delivered for one minute prior to suctioning in Protocol II and for one minute after suctioning in Protocol III. Protocol IV incorporated one minute of hyperinflations prior to and one minute of hyperinflations after suctioning as well as 30 seconds of hyperinflations between the three suction passes. Table 2 contains a summary of the four suction protocols.

In order to establish that changes made in ventilator settings (during the suction protocols) were actually executed properly two standard pieces of I.C.U. equipment were utilized. An Instrumentation Laboratories-O_2 Monitor 404 was attached to all the ventilators used.

Table 2. A description of the four suction protocols

Protocol I	Protocol II	Protocol III	Protocol IV
Increase O_2 (14 min)	Increase O_2 (14 min)	Increase O_2 (14 min)	Increase O_2 (14 min)
Ventilate (1 min)*	Hyperinflate (1 min)***	Ventilate (1 min)	Hyperinflate (1 min)
Suction (15 sec)**	Suction (15 sec)	Suction (15 sec)	Suction (15 sec)
Ventilate (30 sec)	Ventilate (30 sec)	Ventilate (30 sec)	Hyperinflate (30 sec)
Suction (15 sec)	Suction (15 sec)	Suction (15 sec)	Suction (15 sec)
Ventilate (30 sec)	Ventilate (30 sec)	Ventilate (30 sec)	Hyperinflate (30 sec)
Suction (15 sec)	Suction (15 sec)	Suction (15 sec)	Suction (15 sec)
Ventilate (1 min)	Ventilate (1 min)	Hyperinflate (1 min)	Hyperinflate (1 min)
Ventilate	Ventilate	Ventilate	Ventilate

*The term ventilate means that the subject is receiving ventilator breaths at the normal VT.

**The term suction signifies that the subject is disconnected from the ventilator for a total of 15 seconds but only suctioned for 5 seconds during this time.

***The term hyperinflate means that the subject is receiving ventilator breaths 1.5 times the normal VT.

This monitor verified that the FIO_2 was increased by 0.2 fifteen minutes prior to each suction protocol and that it remained at the proper level throughout the suction procedure. A Bournes Adult Ventilation Monitor LS-80 was used to measure the patient's expired VT. This confirmed that hyperinflations were delivered and provided a measure of the actual degree of hyperinflation.

In this study a hyperinflation was defined as mechanical ventilation (via a volume ventilator) at 1.5 times the ventilator setting for tidal volume. If the patient was receiving a VT of 1,000 ml then a hyperinflation would be a 1,500 ml breath delivered by a volume control ventilator. This method was preferred over manual hyperinflations because of the consistency of reproducing the hyperinflation tidal volume delivered to the patient.

Time

At specific times during each protocol testing, data on the dependent variables were recorded (Table 3). For all protocols data were collected before the FIO_2 was increased (pre-baseline), after fourteen minutes of an increased FIO_2 (baseline), and 0 sec., 60 sec., 120 sec., 180 sec. after the third suction pass. Protocols II and IV included one additional time interval, that is, measures were taken after one minute of hyperinflations prior to the first suction pass (post-baseline).

Table 3. Timetable for drawing ABGs and recording $tcPO_2$, ear $Sa0_2$ and heart rate during each of the four suction protocols

Protocol									
I	↑FIO_2 (14 min)	Ventilate* (1 min)	Suction** (15 sec)	Ventilate (30 sec)	***	Suction (15 sec)		Ventilate	
II	↑FIO_2 (14 min)	Hyperinflate◆ (1 min)	Suction (15 sec)	Ventilate (30 sec)	***	Suction (15 sec)		Ventilate	
III	↑FIO_2 (14 min)	Ventilate (1 min)	Suction (15 sec)	Ventilate (30 sec)	***	Suction (15 sec)	Hyperinflate (1 min)	Ventilate	
IV	↑FIO_2 (14 min)	Hyperinflate (1 min)	Suction (15 sec)	Hyperinflate (30 sec)	***	Suction (15 sec)	Hyperinflate (1 min)	Ventilate	
	↑ Pre-Baseline	↑ Baseline	↑ Post-Baseline				↑ Zero Time	↑ 60 sec	↑ 120 sec ↑ 180 sec

x Specifies the time when physiologic parameters are to be recorded and ABGs drawn.

* The term ventilate means that the subject is receiving ventilator breaths at the normal VT.

◆ The term hyperinflate means that the subject is receiving ventilator breaths 1.5 times the normal VT.

** The term suction signifies that the subject is disconnected from the Ventilator for a total of 15 seconds but only suctioned for 5 secs. during this time.

*** Indicates that the preceeding suction (15 sec) and Ventilate/Hyperinflate (30 sec) are to be repeated x 1.

Dependent Variables

It was not clear what dependent measures would be sensitive to Protocol variations therefore a decision was made to use several measures of hypoxemia. The dependent variables included PaO_2, calclated SaO_2, ear oximeter SaO_2, $tcPO_2$ and heart rate.

1. PaO_2, the partial pressure of oxygen in arterial blood, was recorded by machine analysis of arterial blood samples using a Corning 178/pH Blood Gas Analyzer. Arterial blood was taken from the patient's arterial catheter into heparinized plastic syringes. One ml was withdrawn from the catheter and discarded prior to the 0.5 ml sample. Once drawn, a needle was placed on the syringe and the needle was sealed with a rubber stopper. The syringe was subsequently placed on ice and taken to the Blood Gas Laboratory for analysis within one hour of sampling.
2. SaO_2 (calculated), the percentage of oxygen bound to hemoglobin in arterial blood, was calculated from the measured PaO_2 and pH of arterial blood samples using a Corning 178/pH Blood Gas Analyzer.
3. Ear Oximeter SaO_2, the percentage of O_2 bound to hemoglobin in ear capillary blood, was measured by a Hewlett-Packard Ear Oximeter (Model 47201A). This measure was obtained by clipping the ear piece of the oximeter to the pinna of the patient's ear. Light from the ear piece was passed through the ear via fiber optic

bundles and the oximeter, by comparing the relative absorbance of light at several wavelengths, calculated the SaO_2 of the ear capillary blood (Kim, Zolandz & Lerner, 1978).

4. $TcPO_2$, an indirect estimate of PaO_2, was recorded by a Novametrix $tcPO_2$ Monitor using a heated surface electrode (44 °C) secured to the patient's upper right chest below the clavicle. The hyperthermia results in: (1) changes in the structure of the stratum corneum which is thought to allow O_2 to diffuse faster, (2) a shift in the oxyhemoglobin dissociation curve (3) local vasodilatation of dermal capillaries which increases the capillary blood flow so that cutaneous capillary blood O_2 is similar to PaO_2 (Tremper & Shoemaker, 1979).
5. Heart rate was recorded by a Hewlett-Packard bedside cardiac monitor (Model 78342A/78203A) which displayed the mean apical rate and the electrocardiogram.

E. Research Hypotheses

1. Without regard for Time, Protocol IV will result in significantly greater PaO_2 and SaO_2 than Protocols I, II and III.
2. Without regard for Protocol there will be significant fluctuations in PaO_2, SaO_2 and heart rate over Time.
3. There will be a significant interaction between Time and Protocol regarding PaO_2, SaO_2 and heart rate.

4. There will be a significant increase in PaO_2 over all four protocols from pre-baseline to baseline as a result of the increased FIO_2.
5. There will be a significant increase in PaO_2 from baseline to post-baseline in Protocols II and IV as a result of one minute of hyperinflations.
6. In each of the four suction protocols PaO_2 will be highly correlated with $tcPO_2$ over time.
7. In each of the four suction protocols SaO_2 will be highly correlated with ear oximeter readings over time.

F. Data Analysis

To asses hypotheses one through five, the data were analyzed by 2-way ANOVAs with repeated measures. Hypotheses six and seven were addressed using Pearson's product-moment correlation coefficients (r). The level of significance for all statisical tests was set a priori at $p \leq .1$.

IV. Results

The results presented in this chapter are divided into five sections. Sample characteristics are presented in the first section and the outcome measures (PaO_2, SaO_2, heart rate, $tcPO_2$ and ear oximeter SaO_2) are presented in the following four sections.

A. Sample Characteristics

The study population consisted of 19 males and 5 females ranging in age from 18 to 87 years (mean=54, S.D.=20). Table 4 contains a summary of selected sample characteristics including; type of ventilator, tidal volume (VT), backup respiratory frequency, mode of ventilation, FIO_2, size of endotracheal tube (ETT), level of PEEP, and diagnosis.

B. Partial Pressure of Arterial Oxygen

An increased FIO_2 at the beginning of each suction protocol resulted in a significant increase ($p=0.001$) in PaO_2 from pre-baseline 88.02 ± 23.84(S.D.) to baseline 136.09 ± 43.44(S.D.). One minute of hyperinflations given prior to the first suction pass (Protocols II and IV) resulted in a significant increase ($p=0.005$)in PaO_2 from baseline 137.71 ± 43.91(S.D.) to post-baseline 143.02 ± 45.00(S.D.).

Table 4. Characteristics of the sample

ID#	AGE	SEX	VENTILATOR	VT(ml)	FREQUENCY	MODE	FIO_2	ETT(mm)	PEEP	Diagnosis
1	28	M	BearI	700	10	CPAP[1]	.35	8-T[2]	5	M.V.A. **
2	35	M	Servo	700	10	IMV[1]	.30	8-T	5	Cardiac Surgery
3	56	M	MAI	1000	10	CONTROL	.60	8-0[2]	5	M.V.A.
4	31	M	MAI	800	6	IMV	.30	8-T	5	Pancreatitis
5	74	F	MAII	700	10	IMV	.35	7-0	5	Bowel Obstruction
6	25	M	MAII	1000	20	A-C[1]	.30	8-T	3	M.V.A.
7	67	M	BearI	900	10	IMV	.30	8-T	3	Aortic Aneurysm
8	74	M	BearI	800*	10	A-C	.45	8-0	5	Thoractomy
9	49	M	MAI	900	10	A-C	.50	8-0	3	Pulmonary Embolus
10	62	M	BearI	900	12	A-C	.45	8-0	3	Pulmonary Embolus
11	58	F	MAI	900	10	A-C	.40*	7-0	5	Respiratory Arrest
12	29	M	MAI	900	10	A-C	.40	8.5-0	5	M.V.A.
13	18	M	MAI	900	12	A-C	.25	8-0	3	M.V.A.
14	67	M	MAI	1000*	12	A-C	.40*	8-0	5	Bowel Resection
15	74	M	MAI	1000	12	A-C	.50*	8-0	5	Esophagogastrostomy
16	65	M	MAI	900	11	A-C	.30	8-0	0	Myocardial Infarct
17	59	F	MAI	800	20	A-C	.50	7-0	4	Pneumonia
18	73	M	MAI	900	8	A-C	.35	8-0	5	Cancer Right Lung
19	65	M	MAII	900	11	A-C	.45	8-0	5	Ruptured Esophagus
20	24	M	BearI	900	12	A-C	.25	8-N[2]	5	M.V.A.
21	87	F	MAI	800*	9	A-C	.30	8-0	3	Cholecystectomy
22	71	F	MAI	800	10	A-C	.30	7-0	5	Aortic Aneurysm
23	55	M	MAI	900	10	A-C	.30	8.5-0	5	Laparotomy
24	56	M	MAII	1000	8	A-C	.30	7-0	5	Coronary Artery Bypass

* Signifies that there was a change in VT and/or FIO_2 settings between the four protocols.
Subject #8 had a VT of 800ml x 1 and 900ml x 3.
Subject #11 had an FIO_2 of .40 x 2 and .35 x 2.
Subject #14 had a VT of 1,000ml x 2 and 800ml x 2; he also had an FIO_2 of .40 x 2,.35 x 1 and .30 x 1.
Subject #15 had an FIO_2 of .50 x 3 and .55 x 1.
Subject #21 had a VT of 800ml x 1 and 700ml x 3.

[1] CPAP = continuous positive airway pressure
[1] IMV = intermittent mandatory ventilation
[1] A-C = assist-control
[2] T = tracheostomy tube
[2] O = orally intubated
[2] N = Nasally intubated

** motor vehicle accident

With PaO_2 as the dependent measure, a two-way ANOVA determined that there were significant main effects, as hypothesized, for Protocol and Time as well as a significant Protocol x Time interaction (Tables 5 and 6). Pre-baseline and post-baseline PaO_2 values were not included in this analysis.

Table 5. Summary table: Repeated measures 2-way ANOVA for PaO_2

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Ratio	Probability
Protocol Main Effect	10084.50	3	3361.50	3.29	0.026
Error	70538.00	69	1022.29		
Time Main Effect	35260.50	4	8815.13	21.77	0.001
Error	37258.00	92	404.98		
Time x Protocol	1888.50	12	157.38	2.02	0.023
Error	21535.00	276	78.03		

Table 6. Cell means and standard deviations for PaO_2(mm Hg), time x protocol (n=24)

	TIME					
PROTOCOL	Baseline Mean (S.D.)	0 sec. Mean (S.D.)	60 sec. Mean (S.D.)	120 sec. Mean (S.D.)	180 sec. Mean (S.D.)	Row Mean (S.D.)
I	135.46	115.25	129.00	136.38	139.67	131.15
	(41.61)	(43.35)	(46.98)	(47.92)	(47.02)	(45.38)
II	135.17	114.83	128.58	136.04	139.75	130.88
	(44.33)	(44.19)	(44.42)	(45.35)	(45.36)	(44.73)
III	133.50	114.54	135.96	138.08	139.50	132.32
	(45.98)	(41.63)	(43.57)	(42.92)	(43.83)	(43.59)
IV	140.25	125.83	147.50	147.42	148.79	141.96
	(43.85)	(41.87)	(42.05)	(41.10)	(40.36)	(41.85)
Column Mean (S.D.)	136.09	117.62	135.26	139.48	141.93	134.08
	(43.44)	(42.76)	(44.26)	(44.32)	(44.14)	(43.89)

Since the main effects for Time and Protocol were significant, post-hoc Tukey tests (Ferguson, 1981) were conducted for each of the variables to compare pairwise differences. Protocol IV PaO_2 was significantly higher than the other protocols (Table 7). At 0 sec. the PaO_2 was significantly lower than the other time intervals (Table 8).

Table 7. Significant post-hoc Tukey comparisons of row mean PaO_2 values for protocol

	Comparison	q Ratio	(df)	Probability
Protocol	I vs IV	10.81	(4,69)	0.05
	II vs IV	11.08	(4,69)	0.05
	III vs IV	9.64	(4,69)	0.10

Table 8. Significant post-hoc Tukey comparisons of column mean PaO_2 values for time

	Comparison	q Ratio	(df)	Probability
Time	Baseline vs 0 sec.	18.48	(5,92)	0.05
	0 sec. vs 60 sec.	17.65	(5,92)	0.05
	0 sec. vs 120 sec.	21.86	(5,92)	0.05
	0 sec. vs 180 sec.	24.31	(5,92)	0.05

The Protocol x Time interaction is illustrated in Figure 2. The PaO_2 values for the four protocols were not significantly different at baseline(one-way ANOVA). At 0 and 60 sec., however, Protocol IV PaO_2 increased thus accounting for the significant interaction. In addition, Protocols I through III had very similar PaO_2 across time except at 60 sec., when PaO_2 for Protocol III increased. This increase was also responsible, in part, for a significant Protocol x Time interaction.

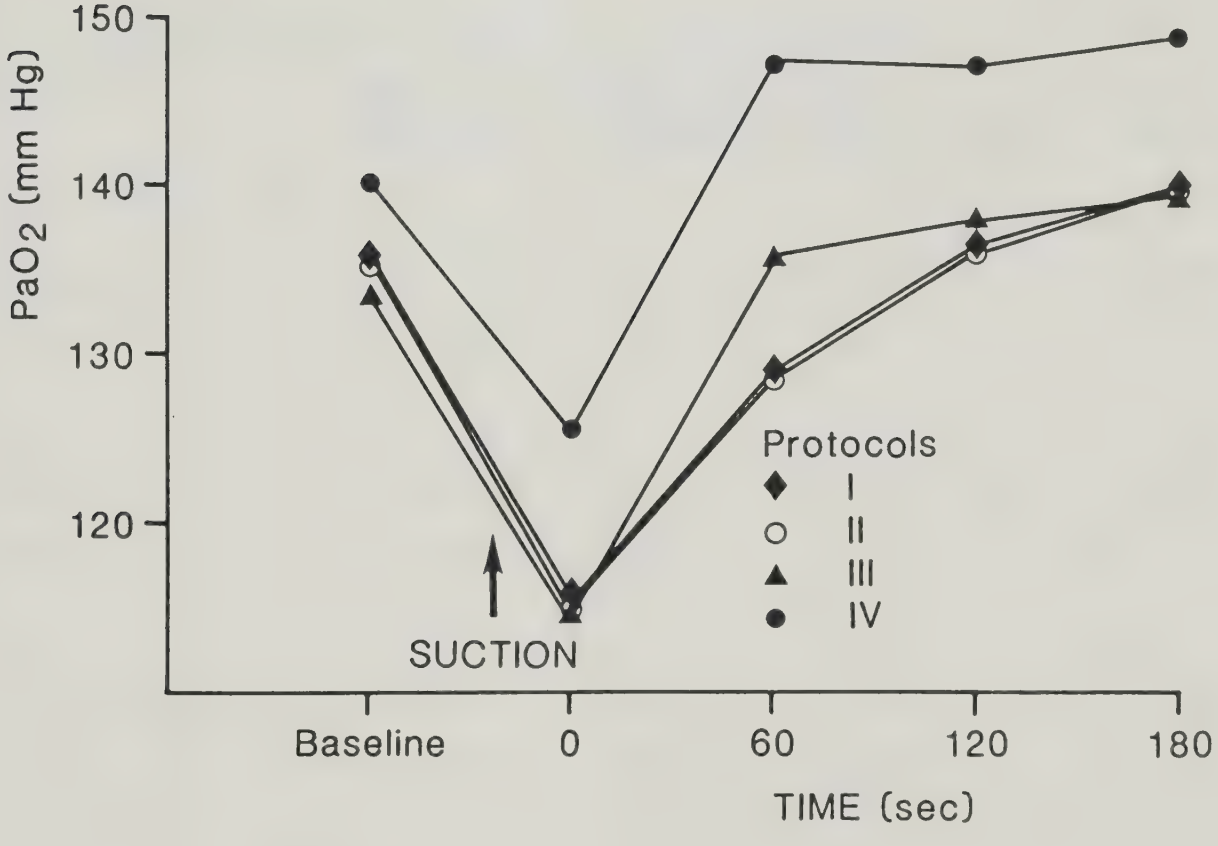


Figure 2. Protocol x time interaction for PaO_2

C. Saturation of Oxygen

The repeated measures 2-way ANOVA for SaO_2 yielded the hypothesized significant main effect for Time and a significant Time x Protocol interaction (Tables 9 and 10). The hypothesized main effect for Protocol however was not supported. Pre-baseline and post-baseline SaO_2 values were not included in this analysis. Since the main effect for Time was significant post-hoc Tukey comparisons were conducted. The SaO_2 at 0 sec. was significantly lower than at all other time intervals (Table 11).

Table 9. Summary table: Repeated measures 2-way ANOVA for SaO_2

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Ratio	Prob-ability
Protocol	33.00	3	11.00	1.49	0.226
Error	511.00	69	7.41		
Time	118.50	4	29.63	14.05	0.001
Error	194.00	92	2.11		
Interaction	15.00	12	1.25	1.97	0.027
Error	175.00	276	0.63		

Table 10. Cell means and standard deviations for SaO_2(%), time x protocol (n=24)

	TIME					
PROTOCOL	Baseline Mean (S.D.)	0 sec. Mean (S.D.)	60 sec. Mean (S.D.)	120 sec. Mean (S.D.)	180 sec. Mean (S.D.)	Row Mean (S.D.)
I	98.46	96.79	97.42	97.92	98.13	97.74
	(1.47)	(3.55)	(3.59)	(2.70)	(2.31)	(2.72)
II	98.00	96.54	97.79	98.04	98.17	97.71
	(2.41)	(3.71)	(2.92)	(2.68)	(2.33)	(2.81)
III	97.96	96.71	97.79	97.96	98.21	97.73
	(2.07)	(3.79)	(2.78)	(2.51)	(2.17)	(2.66)
IV	98.46	97.63	98.58	98.42	98.63	98.34
	(1.69)	(2.98)	(1.82)	(2.10)	(1.95)	(2.10)
Column Mean (S.D.)	98.22	96.92	97.90	98.08	98.28	97.88
	(1.91)	(3.51)	(2.78)	(2.50)	(2.19)	(2.58)

Table 11. Significant post-hoc Tukey comparisons of SaO_2 across time

	Comparison	q Ratio	(df)	Probability
Time	Baseline vs 0 sec.	1.30	(5,96)	0.05
	0 sec. vs 60 sec.	.98	(5,96)	0.05
	0 sec. vs 120 sec.	1.17	(5,96)	0.05
	0 sec. vs 180 sec.	1.36	(5,96)	0.05

The Protocol x Time interaction for SaO_2 is illustrated in Figure 3. At 0 and 60 sec. the SaO_2 during Protocol IV increased thus accounting for the significant interaction. In addition, Protocols I through III had very similar SaO_2 across time except at baseline and 60 sec. The SaO_2 for Protocol I was higher at baseline and lower at 60 sec. compared to Protocols II and III. The decrease in mean SaO_2 at 60 sec. for Protocol I was likely also responsible for the significant Protocol x Time interaction.

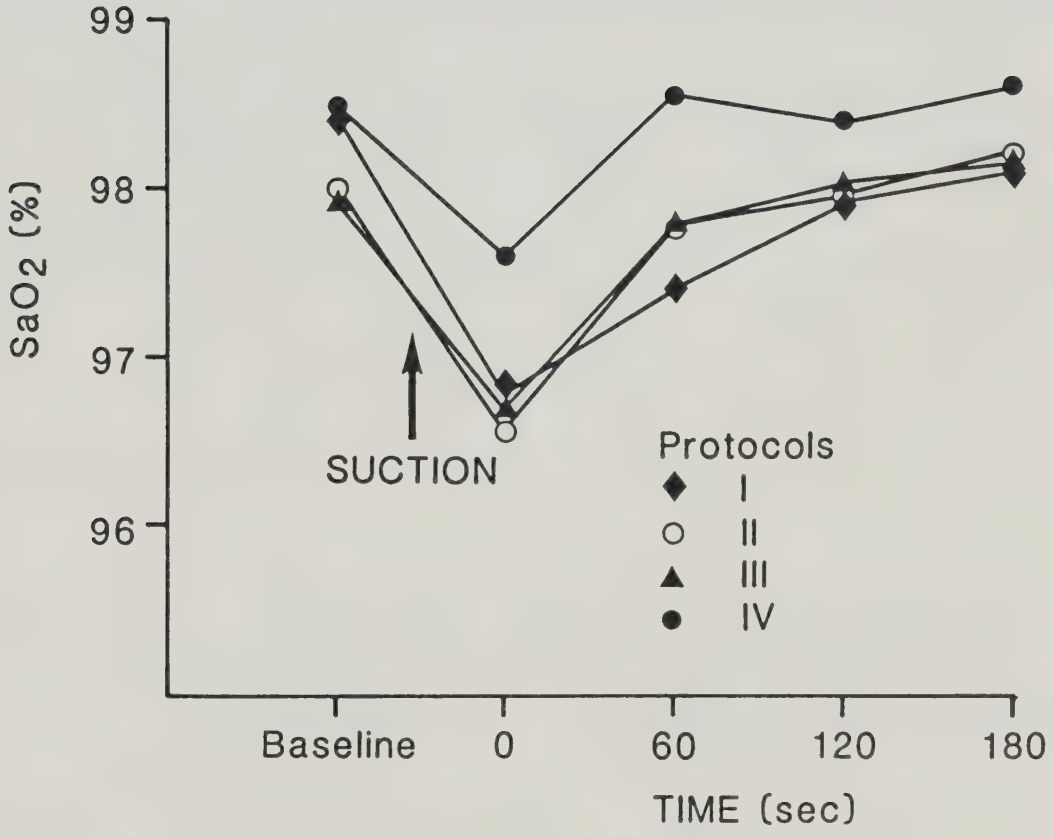


Figure 3. Protocol x time interaction for SaO_2

D. Heart Rate

Results from the analysis of heart rate revealed no significant Protocol effect and no interaction effect. There was however, a significant Time effect (p=0.001). Table 12 contains the mean heart rates across Time. Post-hoc Tukey comparisons revealed that heart rate at 0 sec. was significantly higher than at all other times. Also baseline heart rate was significantly lower than heart rate at 60 sec.(Table 13).

Table 12. Mean heart rate (beats/min.) over time

Baseline Mean (S.D.)	0 sec. Mean (S.D.)	60 sec. Mean (S.D.)	120 sec. Mean (S.D.)	180 sec. Mean (S.D.)
94.21	101.44	98.01	95.39	94.85
(20.30)	(16.90)	(19.91)	(20.83)	(20.43)

Table 13. Significant post-hoc Tukey comparisons of column mean heart rate values for time

	Comparison	q Ratio	(df)	Probability
Time	Baseline vs 0 sec.	7.23	(5,96)	0.05
	Baseline vs 60 sec.	3.80	(5,96)	0.05
	0 sec. vs 60 sec.	3.42	(5,96)	0.05
	0 sec. vs 120 sec.	6.05	(5,96)	0.05
	0 sec. vs 180 sec.	6.58	(5,96)	0.05

E. Transcutaneuos Pressure of Oxygen and Ear Oximetry

Owing to the malfunctioning of the $tcPO_2$ monitor during the study $tcPO_2$ data was not available on two patients. Some data were also missing from another patient because readings from the ear oximeter would not stabilize possibly due to a large amount of ear cartiledge. All other data were collected from these three patients.

Pearson product moment correlations were determined for PaO_2 and $tcPO_2$ (Table 14) and also for SaO_2 and ear oximeter readings (Table 15). This analysis was done across Time, one for each of the four protocols, and the results support the hypothesis that both sets of correlations would be high.

The correlations between PaO_2 and $tcPO_2$ for baseline through 180 sec. ranged from .666 to .870 with the majority around .79. Pre-baseline correlations between PaO_2 and $tcPO_2$ were somewhat lower ranging from -.089 to .647. The mean correlation (excluding pre-baselinevalues) between PaO_2 and $tcPO_2$ was .79 (p=.000).

The correlations between SaO_2 and ear oximeter readings for pre-baseline through 180 sec. ranged from .684 to .908 with the majority of correlations around .84. The pre-baseline correlation for protocol I was .420. Excluding this low value the mean correlation between oximeter SaO_2 and blood SaO_2 was .79 (p=.000).

Table 14. Correlations between $tcPO_2$ and PaO_2 over time for each protocol*

		TIME						
PROTOCOL	N	Pre-Baseline	Baseline	Post-Baseline	0 sec.	60 sec.	120 sec.	180 sec.
I	21	-.089 p=.351	.824	n/a	.821	.870	.870	.845
II	22	.647	.834	.807	.766	.791	.813	.786
III	22	.578	.829	n/a	.796	.799	.792	.820
IV	22	.646	.723	.681	.666	.743	.763	.764

* $p \leq .002$ unless otherwise noted

Table 15. Correlations between SaO_2 and ear oximeter readings over time for each protocol*

	TIME						
PROTOCOL	Pre-Baseline	Baseline	Post-Baseline	0 sec.	60 sec.	120 sec.	180 sec.
I	.420	.684	n/a	.778	.819	.798	.749
	p=.023						
II	.724	.850	.776	.745	.842	.837	.842
III	.786	.885	n/a	.887	.908	.904	.876
IV	.731	.766	.745	.813	.802	.842	.802

For all protocols n=22
* p=.000 unless otherwise noted

V. Discussion, Recommendations and Conclusion

In this section the results of the study will be discussed and implications for nursing practice will be outlined. Recommendations for future research will conclude this chapter.

A. Discussion of Results

The post-hoc Tukey tests revealed that there were no significant differences between Protocols I, II, and III for PaO_2. It made no difference, with respect to the Protocol effect, whether hyperinflations were delivered prior to suctioning (II), after suctioning (III) or omitted altogether (I). The significant main effect can therefore be attributed to Protocol IV which included the administration of hyperinflations before, between and after suctioning.

Physiologically, the administration of hyperinflations prior to suctioning may increase the lung volume, stabilize alveoli and redistribute surfactant thereby causing an increase in PaO_2. In comparing baseline with post-baseline PaO_2 in Protocols II and IV a significant increase in PaO_2 was noted (due to one minute of hyperinflations prior to suctioning). However, this effect was short lived as evidenced by the lack of a difference in PaO_2 after suctioning (0 sec.) between Protocols I (no hyperinflations) and II (hyperinflations prior to suctioning).

The value of hyperinflations delivered after suctioning is supported by the significant increase in PaO_2 at 60 sec. for Protocol III. This increase is important clinically for if a patient is hypoxic, delivery of post-suction hyperinflations may aid in the recovery of PaO_2 to the pre-suction level. Hyperinflations given after suctioning may increase PaO_2 by reinflating collapsed alveoli and increasing the lung volume. A single hyperinflation session post-suctioning (Protocol III) was not enough however to raise PaO_2 to the same level as Protocol IV.

Overall, Protocol IV PaO_2 values were higher than the other three protocols indicating that suctioning places the patient at less risk if this procedure is followed. The delivery of hyperinflations throughout the suction procedure appears to be the cause of the significantly higher PaO_2 for Protocol IV.

A comparison of PaO_2 between Protocols II and IV at 0 sec. seems to indicate that the higher post-suction PaO_2 in Protocol IV is accounted for by the delivery of hyperinflations between each suctioning rather than hyperinflations given prior to suctionings (II).

Physiologically, hyperinflations between each suctioning may serve to keep the alveoli open and re-expand the lungs to their pre-suction volume thereby preventing a cumulative suction effect and minimizing the decrease in PaO_2.

Unlike PaO_2, there was no Protocol effect for SaO_2. This may be due to the fact that SaO_2 is a less sensitive measure than PaO_2, that is, as PaO_2 decreases from 100 mm Hg (or more) to 60 mm Hg there will be very little concomitant change in SaO_2. However, the significant Time x Protocol interaction for SaO_2 (which was very similar in pattern to the PaO_2 interaction) supports the importance of hyperinflations in the suction procedure.

A comparison of Protocols I and II at 0 sec. revealed once again that hyperinflations given before suctioning (II) had no effect in minimizing the fall in SaO_2 post-suction. The higher SaO_2 value for Protocol IV at 0 sec. may be attributed to the administration of hyperinflations between the three suction passes. The increase in SaO_2 at 60 sec. for Protocol IV may be accounted for by the administration of post-suction hyperinflations.

The similar rise in SaO_2 at 60 sec. for Protocols II and III is difficult to explain in terms of hyperinflations as the PaO_2 for Protocol III was higher than that of Protocol II at this time. Perhaps the explanation is that SaO_2 for Protocol I at 60 sec. did not rise as much as the others, that is, the recovery from suctioning was delayed due to the absence of hyperinflations.

The literature on endotracheal suction indicates that the fall in PaO_2 and SaO_2 post-suctioning is due to a combination of factors, namely; (1) the removal of oxygen from the lungs and the resulting decrease in lung volume,

(2) the flow of room air into the lungs during suctioning, (3) respiratory reflexes (tracheobronchoconstriction), and (4) atelectasis caused by large negative airway pressure.

Urban and Weitzner (1969) documented a mean 35 mm Hg fall in PaO_2 immediately after suctioning and Skelley, Deeren and Powaser (1980) reported a mean 33 mm Hg fall in PaO_2 thirty seconds after suctioning. The drop in PaO_2 immediately after suction (0 sec.) in this study was a mean of 18.5 mm Hg.

Perhaps the following procedural differences account for the larger drop in PaO_2 reported in the aforementioned studies; (1) Patients in these studies were not preoxygenated with an increased FIO_2 therefore the fall in PaO_2 would be greater than in the current study. (2) These researchers applied continuous suction for fifteen seconds as opposed to the three, five second suctionings (with thirty seconds of ventilation between each suctioning) used in this study.

As with PaO_2 there was a significant drop in SaO_2 at 0 sec. due to suctioning. This would be expected in view of the drop in PaO_2 occurring at this time. Heart rate however, increased significantly immediately after suctioning (0 sec.). This may also be explained in terms of the body's response to a decrease in PaO_2, that is, the release of epinephrine.

In conducting this study it was important to control for the effects of oxygen. The findings indicate that

increasing the FIO_2 by 0.2 prior to suctioning (pre-baseline) resulted in a significant increase in PaO_2 at baseline. Such an increase was likely beneficial to the patients as it may have maintained a higher PaO_2 post-suctioning.

The increase in PaO_2 associated with the 0.2 increase in FIO_2 will fluctuate however, depending on the patient's respiratory status. A patient with healthy lungs on an FIO_2 of 0.3 will likely have a greater increase in PaO_2 when the FIO_2 is raised by 0.2 than another patient who is in respiratory failure and on an FIO_2 of 0.8.

In this study patients were given fourteen minutes of increased FIO_2 at the beginning of each suction protocol. It is not known whether a shorter or longer period of preoxgenation would have produced a similar baseline increase in PaO_2. In a study by Tremper and Schoemaker (1981) PaO_2 reached 95% of its equilibrium value two minutes after the FIO_2 was changed. This data suggest that a shorter period of preoxygenation may have been sufficient.

Finally, the correlation between PaO_2 and $tcPO_2$ (Novametrix monitor) was .79. This finding is consistent with the correlation of .75 reported by Rafferty, Schacter and Barash (1980). The correlation between calculated SaO_2 and ear oximeter SaO_2 (Hewlett-Packard) was also .79. This finding is much lower than the .97 reported by Saunders, Powles, and Rebuck (1976). The major advantage of these monitors is that they provide a noninvasive, continuous

means of monitoring arterial oxygenation in acutely ill patients. Use of these devices in I.C.U. may decrease the number of blood gas samples necessary for routine clinical care.

B. Nursing Implications

The results of this study clearly demonstrate that hyperinflations delivered throughout the suction procedure minimized the fall in PaO_2 associated with suctioning. Based on these findings it is recommended that routine ICU suction procedures executed on ventilated patients (VT ≤ 1,000ml, no PEEP, on Assist-Control mode) be modified to include the administration of hyperinflations after suctioning as well as between each suction pass. In this study hyperinflations prior to suctioning were not shown to be of value with respect to minimizing the fall in PaO_2 or SaO_2 post-suctioning.

The findings of this study support the current practice of increasing the FIO_2 several minutes prior to suctioning as this appears to protect the patient from a disastrously low PaO_2 post-suction.

The following recommendations regarding time were not investigated in this study but emerge from the author's experience with the suction procedure. First, patients should be given time to recover from each suctioning. In this study thirty seconds of mechanical ventilation was provided between the three suction passes. To the nurse at

the bedside this pause may seem interminable, however, this would appear to be a reasonable time to wait. Second, the length of time the paitent is off the ventilator, the duration of actual suction and the number of suction passes should be kept to a minimum as these factors would appear to affect the change in PaO_2 associated with suctioning.

C. Recommendations for Further Study

This study was conducted: (1) on a small sample of acutely ill ventilated adults, with varying illnesses, who met certain criteria ($FIO_2 \leq 0.6$, VT ≤ 1,000 ml, no PEEP, on Assist-Control mode), and (2) using suction protocols which included 14 minutes of preoxygenation and excluded instillations. These limitations prompt the following recommendations for future research.

1. This study should be conducted in various critical care settings to establish the validity of the findings.
2. Controlled clinical studies should be conducted to compare the effects of endotracheal suction and hyperinflation under the following conditions:
    a. different ventilatory modes (assist-control versus intermittent mandatory ventilation),
    b. varying levels of PEEP,
    c. varying lengths of preoxygenation,
    d. incorporating an instillation into the procedure, and
    e. specific disease states for instance pulmonary

embolus verus pneumonia.

D. Conclusions

Suctioning is necessary to clear secretions and maintain airway patency in intubated patients. The removal of oxygen from the tracheobronchial tree during suctioning leads to alveolar hypoxia and hypoxemia post-suctioning and an extended period of hypoxemia places the patient at risk for fatal cardiac arrhythmias.

In this study preoxygenation for 14 minutes with an increased FIO_2 and hyperinflations delivered by a volume ventilator before, between, and after suctioning minimized the decrease in PaO_2 associated with endotracheal suctioning.

VI. REFERENCES

Adams, A. & Hahn, C. Principles and practice of blood-gas analysis. Great Britain: W.S. Cowell Ltd., 1979.

Adlkofer, R. & Powaser, M. The effect of endotracheal subtioning on arterial blood gases in patients after cardiac surgery. Heart and Lung, Nov.-Dec., 1978, 5(6), 1011-1014.

Ayres, S. & Grace, W. Inappropriate ventilation and hypoxemia as causes of cardiac arrhythmias. American Journal of Medicine, April, 1969, 46, 495-505.

Balsys, A., Jones, R., Man, S., & Wells, A. Effects of sighs and different tidal volumes on compliance, functional residual capacity and arterial oxygen tension in normal and hypoxemic dogs. Critical Care Medicine, Nov., 1980, 8, 641-645.

Barnes, C., Asonye, V., & Vidyasahar, D. The effects of bronchopulmonary hygiene on $PtcO_2$ values in critically ill neonates. Critical Care Medicine, Dec., 1981, 9(12), 819-822.

Belling, D., Kelley, R., & Simon, R. Use of the swivel adaptor aperture during suctioning to prevent hypoxemia in the mechanically ventilated patient. Heart and Lung, Mar.-Apr., 1978, 7, 320-322.

Benson, M. & Matheny, R. Washout volumes of three commonly used ventilators. Respiratory Care, 1978, 23, 74.

Berman, I & Stahl, W. Prevention of hypoxic complications during endotracheal suctioning. Surgery, Apr., 1968, 63(4), 586-587.

Boba, A., Cincotti, J., Piazza, T., & Landmesser, C. The effects of apnea, endotracheal suction, and oxygen insufflation, alone and in combination, upon arterial oxygen saturation in anesthetized patients. Journal of Laboratory and Clinical Medicine, 1959, 53, 680-685.

Bodai, B. A means of suctioning without cardiopulmonary depression. Heart and Lung, Mar., 1982, 11(2), 172-176.

Boutros, A. Arterial blood oxygenation during and after endotracheal suctioning in the apneic patient. Anesthesiology, Feb., 1970, 32, 114-118.

Brandstater, B. & Muallem, M. Atelectasis following tracheal suction in infants. Anesthesiology, 1969, 31,

468.

Brannin, P. Oxygen therapy and measures of bronchial hygiene. Nursing Clinics of North America, Mar., 1974, 9(1), 111-121.

Campbell,D., & Stanley, J. Experimental and quasi-experimental designs for research. Chicago: Rand McNally College Publishing Co., 1963.

Capen, R., Latham, L., & Wagner, W. Diffusing capacity of the lung during hypoxia: Role of capillary recruitment. Journal of Applied Physiology, Jan., 1981, 50(1), 165-171.

Christenson, L. Experimental Methodology. Boston: Allyn & Bacon, Inc., 1980.

Comroe, J. Physiology of respiration. Chicago: Year Book Medical Publishers Inc., 1974.

Connors, A., Hammon, W., Martin R., & Rogers, R. Chest physical therapy. The immediate effect on oxygenation in acutely ill patients. Chest, Oct., 1980, 78(4), 559-564.

Corbett, J., Kerr, J., & Prys-Roberts, C. Cardiovascular responses to aspiration of secretions from the respiratory tract in man. Journal of Physiology, 1968, 201, 51P-51P.

Deeren, S. & Powaser, M. The influence of continuous versus intermittent endotracheal suctioning on arterial oxygen tension. American Review of Respiratory Disease, Apr., 1979, 119(4), 188.

Demers, R. Complications of endotracheal suctioning procedures. Respiratory Care, Apr., 1982, 27(4), 453-457.

Demers, R. & Saklad, M. Minimizing the harmful effects of mechanical aspiration. Heart and Lung, July-Aug., 1973, 2, 542-545.

Downes, J., Wilson, J., & Goodson, D. Apnea, suction, and hyperventilation: Effect on arterial oxygen saturation. Anesthesiology, Jan.-Feb., 1961, 22, 29-33.

Dripps, R. & Comroe, J. The effect of the inhalation of high and low oxygen concentrations on respiration, pulse rate, ballistocardigram and arterial oxygen saturation (oximeter) of normal individuals. The American Journal of Physiology, May, 1947, 149, 277-291.

Ehrhart, I., Hofman, W., & Loveland, S. Effects of endotracheal suction versus apnea during interruption of intermittent or continuous positive pressure ventilation. Critical Care Medicine, June, 1981, 9, 464-467.

Fell, T. & Cheney, F. Prevention of hypoxia during endotracheal suction. Annals of Surgery, July, 1971, 179, 24-28.

Ferguson, G. Statistical analysis in psychology and education. New York: Mcgraw-Hill Book Co., 1981.

Fletcher, G. & Barber, J. Effect of sampling technique on the determination of PaO_2 during oxygen breathing. Journal of Applied Physiology, 1966, 21, 463-468.

Fletcher, G. & Barber, J. Lung mechanics and physiologic shunt during spontaneous breathing in normal subjects. Anesthesiology, 1966, 27, 638-647.

Grossbach-Landis, I. & McLane, M. Tracheal suctioning: A tool for evaluation and learning needs assessment. Nursing Research, July-Aug., 1979, 28, 237-242.

Guyton, A. Basic human physiology: Normal function and mechanisms of disease. Toronto: W.B. Saunders Co., 1971.

Harken, A. A routine for safe, effective, endotracheal suctioning. The American Surgeon, July, 1975, 41, 398-404.

Hedley-Whyte, J., Laver, M., & Bendixen, H. Effect of changes in tidal ventilation on physiologic shunting. American Journal of Physiology, 1964, 206, 891-897.

Hilding A. Time-lapse relation to changes in the respiratory epithelium after minimal trauma. Acta Otolaryngologica (Stockholm), 1964, 57, 352-366.

Hunter, P. Bedside monitoring of respiratory function. Nursing Clinics of North America, June, 1981, 6, 211-224.

Jacobs, M. Sources of measurement error in noninvasive electronic instrumentation. Nursing Clinics of North America, Dec., 1978, 13(4), 573-587.

Jacquette, G. To reduce hazards of tracheal suctioning. American Journal of Nursing, Dec., 1971, 71(12), 2362-2364.

Jung, R. & Gottlieb, L. Comparison of tracheobronchial

suction catheters in humans, Chest, Feb., 1976, 6 (2), 179-181.

Jung, R. & Newman, J. Minimizing hypoxia during endotracheal airway care. Heart and Lung, May-June, 1982, 11(3), 208-212.

Katz, A., Michelson, E., Stawicki, J., & Holford, F. Cardiac arrhythmias: Frequency during fiberoptic bronchoscopy and correlation with hypoxemia. Archives of Internal Medicine, April, 1981, 141, 603-606.

Keown, K. Cardiac arrest during therapeutic tracheal suction. Anesthesia and Analgesia, Nov.-Dec., 1960, 39, 568-569.

Kergin, F., Bean, D. & Paul, W. Anoxia during intrathoracic operations. A preliminary report. Journal of Thoracic Surgery, 1948, 17, 709-711.

Keszler, H. Tracheobronchial toilet without cardiorespiratory impairment. Critical Care Medicine, May, 1980, 8, 298-301.

Kim, B., Zolandz, R., & Lerner, S. Techniques for continuous analysis of arterial blood gases. Anesthesiology Review, June, 1978, 5(6), 19-26.

Kuzenski, B. Effect of negative pressure on tracheobronchial trauma. Nursing Research, July-Aug., 1978, 27, 260-263.

Landa, J., Chapman, G., & Sackner, M. Effects of suctioning on mucociliary transport. Chest, Feb., 1980, 77, 202-207.

Langrehr, A., Washburn, S., & Guthrie, M. Oxygen insufflation during endotracheal suctioning. Heart and Lung, Nov.-Dec., 1981, 10(6), 1028-1036.

Link, W., Spaeth, E., Wahle, W., Penny, W., & Glover, J. The influence of suction catheter tip design on tracheobronchial trauma and fluid aspiration efficiency. Anesthesia and Analgesia, Mar.-Apr., 1976, 55, 290-297.

Marx, G., Steen, S., & Arkins, R. Edotracheal suction and death. New York State Journal of Medicine, 1968, 68, 565.

Mountcastle, V. Medical physiology(Volume 2). St. Louis: C.V. Mosby Co., 1974.

Nadel, A. & Widdicombe, J. Reflex effects of upper airway irritation on total lung resistance and blood pressure.

Journal of Applied Physiology, Nov., 1962, 17, 861-865.

Naigow, D., & Powaser, M. The effect of different endotracheal suction procedures on arterial blood gases in a controlled experimental model. Heart and Lung, Sept.-Oct., 1977, 6, 808-816.

Nie, N., Hull, C., Jenkins, J. Steinbrenner, K., & Bent, D. SPSS statistical package for the social sciences (2nd edition). New York: McGraw-Hill Book Co., 1975.

Peabody, J., Willis, M., Gregory, G., Tooley, W., & Lucey, J. Clinical limitations and advantages of transcutaneous oxygen electrodes. Acta Anaesthesia Scandinavia, 1978, Supplement 68, 76-82.

Petty, T. Intensive and rehabilitative respiratory care. Philadelphia: Lea and Febiger, 1976.

Pirlo, A., Benumof, J., & Trousdale, F. Potentiation of lobar hypoxic pulmonary vasoconstriction by intermittent hypoxia in doge. Anesthesiology, Sept., 1981, 55, 226-230.

Powaser, M., Langrehr, E., & Cambell, S. Oxygen insufflation during endotracheal suctioning as a method to prevent suction induced hypoxemia in experimental studies using dogs and post cardiac surgery patients. American Review of Respiratory Disease, April, 1979, 119(4), 192.

Pratter, M. & Irwin, R. The effect of transtracheal aspiration on cardiac rate and rhythm. Chest, Oct., 1981, 80(4), 439-441.

Rafferty, T., Schacter, N., & Barash, P. Transcutaneous oxygen and carbon dioxide monitoring in adult surgical patients. Critical Care Medicine, Apr., 1980, 8(4), 269.

Robertson, K. & Guzzetta, C. Arterial blood-gas interpretations in the respiratory intensive-care unit. Heart and Lung, Mar.-Apr., 1976, 5(2), 256-260.

Rokosky, J. Assessment of the individual with altered respiratory function. Nursing Clinics of North America, June, 1981, 16, 195-206.

Rosen, M. & Hillard, E. The use of suction in clinical medicne. British Journal of Anaesthesia, 1960, 32, 486-503.

Rosen, M. & Hillard, E. The effects of negative pressure during tracheal suction. Anesthesia and Analgesia,

Jan.-Feb., 1962, 141(1), 50-55.

Rux, M. & Powaser, M. The effect of apnea and three levels of negative airway pressure on the fall in arterial oxygen tension produced by endotracheal suctioning in dogs. American Review of Respiratory Disease, April, 1979, 119(4), 193.

Sackner, M., Landa, J., Greeneltch, N., & Robinson, M. Pathogenesis and prevention of tracheobronchial damage with suction procedures. Chest, Sept., 1973, 64(3), 284-290.

Saunders, N., Powles, A., & Rebuck, A. Ear oximetry: Accuracy and practicability in the assessment of arterial oxygenation. American Review of Respiratory Disease, 1976, 113, 745-749.

Schumacker, H. & Hampton, L. Sudden death occurring immediately after operation in patientts with cardiac disease with particular reference to the role of aspiration through the endotracheal tube and extubation. Journal of Thoracic Surgery, 1951, 21, 48.

Severinghaus, J. Current trends in continuous blood gas monitoring. Biotelemetry Patient Monitoring, 1979, 6, 9-15.

Severinghaus, J. Transcutaneous blood gas analysis. Respiratory Care, Feb., 1982, 27(2), 152-159.

Shim, C., Fine, N., Fernandez, R., & Williams, M. Cardiac arrhythmias resulting from tracheal suctioning. Annals of Internal Medicine, Dec., 1969, 71(8), 1149-1153.

Simbruner, G., Coradello, H., Fodor, M., Havelec, L., Lubec, G., & Pollak, A. Effect of tracheal suction on oxygenation, circulation, and lung mechanics in newborn infants. Archives of Disease in Childhood, 1981 56, 326-330.

Skelley, B., Deeren, S., & Powaser, M. The effectiveness of two preoxygenation methods to prevent endotracheal suction-induced hypoxemia. Heart and Lung, Mar.-Apr., 1980, 9(2), 316-323.

Sloan, H. The vagus nerve in cardiac arrest. The effect of hypercapnia, hypoxia and asphyxia on reflex inhibition of the heart. Surgery Gynecology and Obstetrics, Sept., 1950, 91(3), 257-264.

Spoerel, W. & Chan, C. Jet ventilation for tracheobronchial suction. Anesthesiology, Oct., 1976, 45, 450-452.

Taylor, A. & Waters, H. Arterial oxygen tensions following endotracheal suction on IPPV. Anesthesia, July, 1971, 26, 289-293.

Thambiran, A. & Ripley, S. Observations on tracheal trauma following suction: An experimental study. British Journal of Anaesthesia, June, 1966, 38, 459-462.

Tomori, Z. & Widdicombe, J. Muscular, bronchomotor and cardiovascular reflexes elicited by mechanical stimulation of the respiratory tract. Journal of Physiology, 1969, 200, 25-49.

Tremper, K. & Shoemaker, W. Transcutaneous PO_2 monitoring useful in adults too. Critical Care Monitor, 1979, 7, 530-535.

Tremper, K. & Shoemaker, W. Transcutaneous oxygen monitoring of critically ill adults, with and without low flow shock. Critical Care Medicine, Oct., 1981, 9(10), 706-709.

Tremper, K., Waxman, K., Bowman, R., Shippy, C., & Shoemaker, C. Transcutaneous oxygen monitoring during respiratory failure, cardiac decompensation, cardiac arrest and CPR. Critical Care Medicine, Apr., 1980, 8(4), 269.

Tremper, K., Waxman, K., Greene, R., Shippy, C., Shoemaker, C. Use of transcutaneous oxygen sensor to optimize PEEP. Critical Care Medicine, Apr., 1980, 8(4), 269.

Urban, B. & Weitzner, S. Avoidance of hypoxemia during endotracheal suction. Anesthesiology, Nov., 1969, 31(5), 473-475.

Weitzner, S., King, B. & Ikezono, E. The rate of arterial oxygen desaturation during apnea in humans. Anesthesiology, 1959, 20(5), 624-627.

Woodburne, C. & Powaser, M. Mechanisms responsible for the sustained fall in arterial oxygen tension after endotracheal suctioning in dogs. Nursing Research, Sept.-Oct., 1980, 29, 312-316.

VII. Appendix I

Consent Form

Research Consent Form

This is to certify that I _____ (relation to patient if relative signing), have given consent for _____ to participate in a research project being conducted by Susan MacKinnon-Kesler, a registered nurse, in the intensive care unit (ICU). I understand that Susan MacKinnon-Kesler: (1) is investigating four common suctioning routines to establish which one is most beneficial to the patient and (2) has secured permission from Dr. E.G. King, Director of ICU, to conduct this study. I also understand that:

a. The suctioning routines to be investigated differ in only one respect from the usual ICU procedure. The difference is that participants will receive some "bigger breaths" from their ventilator before, during and/or after suctioning.
b. The use of "bigger breaths" is common practice in intensive care units and does not cause pain or expose the participant to undue risks.
c. Participants will be studied during four suctionings in a 24 hour period and each time a different suctioning routine will be used.
d. Participants will have about three tablespoons of blood taken from their arterial line during the study period. This is a painless way to draw blood.
e. Participants will have two oxygen monitors attached to them during each of the four study suctionings. These monitors will cause no discomfort to the participant as one monitor will be clipped onto the ear lobe, like an earring, and the other - a small round adhesive pad - will be attached to the chest.
f. The names of the study participants will be kept confidential and will not appear in any research report.
g. Participants will be free to withdraw from the study at any time with no consequences.
h. Participants may not directly benefit from participating in this project.

_____

Signature Investigator Date

_____

Witness